Passive Smoking and the Seasonal Difference of Severity of Asthma in Children*

Andrew B. Murray, M.B.;† and Brenda J. Morrison, Ph.D.‡

To learn whether asthmatic children are affected by passive smoking, we studied 240 unselected consecutively referred asthmatic subjects, aged 7 to 17 years. To discover whether children of smokers are affected more severely during the cold, wet season, when windows are closed and children are indoors, than during the warm, dry season, when houses are well ventilated and children spend more time outdoors, we compared lung function tests recorded during the two seasons. If seen during the cold, wet season, children of smoking mothers compared with those of nonsmoking mothers had a lower FEV,% (74 vs 86, p=.00), FEF25-75 percent (56 vs 75, p=.00) and PC_m histamine (0.85 vs 1.95, p=.01). There was a highly significant correlation between the number of cigarettes the mother smoked in the house and each of these lung function test results, indicating a dose-response relationship. Those seen during the warm, dry season, by contrast, did not have lower mean spirometric test results if their mothers were smokers than if nonsmokers, and there was no correlation between the number of cigarettes the mother smoked in the house and the result of any lung function test. Our results strongly support the hypothesis that cigarette smoke from the mother aggravates her child's asthma. (*Chest* 1988; 94:701-05)

Several studies have reported that children who are exposed to their parents' cigarette smoke are more likely than children of nonsmoking parents to wheeze and to have decreased spirometric test results.[1-4] Although this decrease in pulmonary function is thought to result from passive smoking, a causal relationship is not universally accepted. Doubt remains because some surveys find no difference in

For editorial comment see page 675

spirometric results between the children whose parents smoke and those whose parents do not smoke.[7-9]

Weiss et al[10] suggested a plausible explanation for the failure of these latter studies to show an association between parental smoking and impaired pulmonary function. It is that the surveys were conducted in Arizona, a place where the weather is warm and dry, where children spend more time outdoors, and where ventilation rates of houses are high. As a result, the amount of passive smoking may be less in smoking parents' children who live in Arizona than in those who live in a cold, wet area.

The indoor level of smoke appears to be low in Arizona, even when there is a smoker in the house. In a study carried out in Tucson, Lebowitz[9] found that the indoor concentration of carbon monoxide (CO), an indicator of the smoke level, was comparable to the outdoor concentration, even though a smoker was present. When a building is well ventilated, CO from cigarette smoke is rapidly removed, but when poorly ventilated, as is the case during the heating season in colder areas, the CO concentration increases.[11] The reason for reducing the ventilation rate in cold weather is to conserve energy.[12] The consequence is that in a heated building the air is recirculated and there is an accumulation of cigarette smoke, shown by high concentration of both CO[13] and the mean mass respirable particulate (MRP), another indicator of the amount of cigarette smoke present.[14,15] Dockery and Spengler[16] found that smoke from one pack of cigarettes raised the MRP level by approximately 42 μg/cu m when the air in the building was being recirculated, but only by 18 μg/cu m when the air was not being recirculated.

If passive smoking is greater in cold, wet weather than in warm, dry weather, and if the smoke impairs lung function, we would expect children of smoking mothers in Vancouver to be more severely affected in the cold, wet season (October through May), than in the warm, dry season (June through September; Fig 1).[16] During this warm period, windows and doors are left open, a practice that rapidly changes the indoor air.[16]

Asthmatic children are appropriate subjects for a study to find whether this seasonal difference is present, because their bronchi seem to be more sensitive to the effects of smoke than are those of normal children. The difference in spirometric test results between children of nonsmokers and smokers reported in asthmatic subjects[17] is much greater than reported in representative groups of schoolchildren.[1-4] In such a study the mothers' smoking habits are particularly important, since children have greater

*From the Department of Paediatrics, British Columbia Children's Hospital, and the Department of Health Care and Epidemiology, University of British Columbia, Vancouver, Canada.
†Professor of Paediatrics.
‡Associate Professor of Health Care and Epidemiology.
Supported in part by a grant from the British Columbia Lung Association.
Manuscript received November 13; revision accepted March 21.
Reprint requests: Dr Murray, 4480 Oak Street, Vancouver, B.C., Canada V6H 3V4

2023380676

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

exposure to her smoke than to smoke from the father, judged by cotinine levels in the children's saliva. The level is significantly higher if the smoker in the family is the mother rather than the father.[10]

The present study had two purposes: (1) to confirm that asthmatic children of mothers who smoke do have lower lung function test results than those of mothers who do not smoke, and (2) to find whether these children of smokers are affected more severely in the cold, wet season than in the warm, dry season.

Material and Methods

Population

The series included every child with asthma or a wheezy chest who was aged 7 to 17 years and had been referred to one of us (ABM) at the Children's Hospital in Vancouver.

There were 247 subjects. Of these, 160 were being seen for the first time, and 87 had been examined on a previous occasion, they were being reassessed at the request of their primary care physicians. For both new and old patients only the data recorded at the initial visit during the period Nov. 1, 1983, to May 31, 1986, were used in the study.

Because of a long waiting list, an average period of four months elapsed between the date of referral and the day of the child's visit. As parents tend to forget instructions during such a long interval, every attempt was made to contact them by telephone 48 hours before the appointment. They were asked which medications the child was receiving and were instructed to discontinue those that might influence the bronchial challenge test.

Questionnaire

A trained interviewer put standardized questions to the patients and to the accompanying adult who, in 97 percent of cases, was a parent. All were asked how long the child had had the asthma or wheezing, whether the child had suffered from a cold or respiratory infection during the preceding two weeks,[11,12] and whether the child had received recent medication—ie, corticosteroid or antihistamine medication in the past 48 hours, theophylline in the previous 24 hours, or sodium cromoglycate or bronchodilator in the eight hours before the appointment.

Those who were being seen for the first time were also asked the following questions: whether a gas stove was used for cooking, the type of central heating in their house, whether they owned any furred household pets, and whether either of the parents or any of the siblings had ever had asthma. These items of information had also been ascertained at a previous visit for those referred for reassessment, but because conditions could have changed since the original enquiry, these old data were not used in the present study.

When the above questions had been completed, the parents or accompanying adults were asked how many cigarettes the mother and father smoked per day, both the total number and the number smoked while in the house.

The child was asked privately whether he or she smoked. Of the four who admitted to being smokers, two were children of a nonsmoking mother, and two were children of a smoking mother. Because the purpose of this study was to determine the effect of passive smoking only, these four subjects were excluded.

Forced Expiratory Spirogram

Forced expiratory maneuvers were performed until there were three in which the forced vital capacity (FVC) agreed within 5 percent. This was achieved within five efforts on all except three children, all aged seven years, who were too uncoordinated or too uncooperative to perform a forced expiratory maneuver. These children were eliminated from the series. In the remaining 240 subjects the tracing that had the greatest sum of FVC and forced expiratory volume at one second (FEV_1) was used for all measurements. The FEV_1 and the forced expiratory flow rate during the middle half of the FVC (FEF25-75) were expressed as a percentage of predicted mean for age, sex, and height.[13]

The spirogram was recorded with a Pulmonaire (Jones Medical Instrument Co) waterless spirometer that was calibrated weekly with a known volume of carbon dioxide (CO_2) discharged at a standard velocity from a calibrator instrument.

The results of the tests were analyzed and printed by a Datamatic (Jones Medical Instrument Co) computer connected to the spirometer.

Bronchial Reactivity to Histamine

A bronchial challenge test was performed on all except the following subjects: those who had taken recent medication, as defined above, those who reported a cold or respiratory infection within the preceding two weeks, or those who had an FEV_1 less than 80 percent predicted, or was below 1 L in volume.

A modification[14] of the protocol of Cockcroft et al[15] was used to find the threshold dose of histamine that would result in a fall of 20 percent in the FEV_1. After baseline spirometric measurements had been recorded the patient held a loosely fitting plastic mask to his face, inhaling an aerosol generated by air flowing at 5 L/min through a Wright nebulizer. The volume of solution aerosolized was 0.2 ml/min. Doubling concentrations of histamine acid phosphate were given until the strongest concentration, 8 mg/ml, was reached. Children whose FEV_1 did not decrease by 20 percent when this concentration was administered were deemed, for the purpose of calculating the PC_{20}, to respond to double that concentration, ie, 16 mg/ml histamine acid phosphate. There were 13 such subjects. The mothers of 12 of them were nonsmokers, and the mother of the remaining one was a smoker.

Using a standard method,[16] skin prick tests were performed on all subjects with the following materials: a negative (saline) and a positive (histamine) control solution, extracts of 1 percent *Dermatophagoides farinae* and 1 percent *D pteronyssinus* (Bencard Division of Beecham Laboratories), dog and cat hair (Hollister), and alder, birch, and mixed grass pollens (Greer Lab).

The diameter of each resulting wheal was measured. If any wheal was 2 mm greater than that of the negative control solution, the test was considered positive.

The spirometric, bronchial challenge, and skin tests were performed by a technician who was unaware of the family's smoking habits.

Season

Spirometric and bronchial challenge test results in the children seen between June and September, the warm, dry season, were compared with those recorded between October and May, the cold, wet season in Vancouver[17] (Fig 1).

From October through May there are, on average, no degree days above 18°C.[18] A "degree day" is defined as a measure of the departure for a day from some reference temperature. The reference temperature chosen for this study (18°C) is the one used for residential space heating needs.[18] Heat is required on the days when there is a negative departure from 18°C and no heat on the days when there is a positive departure. From early October until the end of May, therefore, windows and doors are closed every day to conserve heat.

Korsgaard[19] found that windows are left open for 20 hours a day in June and July but only for one hour a day in February and March. Under such circumstances the ventilation rate of the house is decreased, and the cigarette smoke generated in the house reaches high levels.[11,18,19]

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000
2023380677

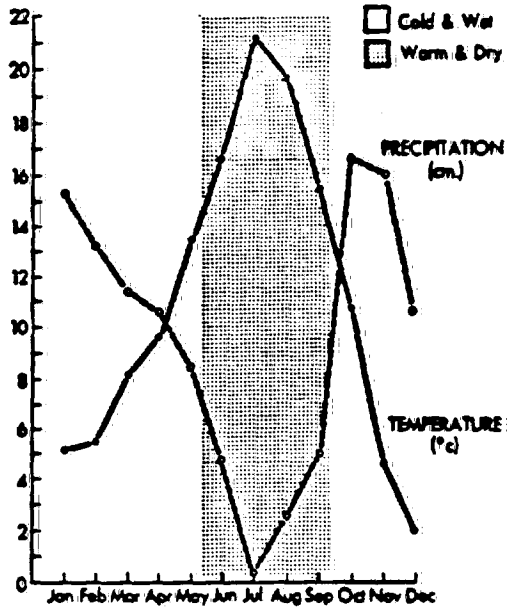

FIGURE 1. The mean monthly temperature and the mean monthly precipitation in Vancouver between November 1983 and June 1986.

From June through September there are degree days above 18°C in every month, and on these days ventilation is unrestricted, as air-conditioning is unnecessary for comfort. The mean maximum temperature in Vancouver in July and August is only 21°C.[16] Of our patients in the Vancouver area, four had air-conditioning in the parents' bedroom, but none had air-conditioning for the whole house. Two patients from cities in the interior of the province had house-wide (central) air-conditioning, the family members in these houses were all nonsmokers.

Statistical Analysis

After elimination of the four children who were themselves smokers and the three who could or would not perform an acceptable spirogram, 240 subjects remained: 183 had mothers who were nonsmokers, 56 had mothers who were smokers, and the smoking status of one mother was unknown.

For statistical analysis, differences were tested by Student's *t* test for those variables on a continuous scale, or a scale approximating it, and by χ^2 for those which were frequencies. Pearson's product-moment and point biserial correlation coefficients, as appropriate, were calculated to assess association. A logarithmic transformation was applied to the number of cigarettes smoked by each of the parents, the PC_{20}, and to other variables that were skewed to the right. Multiple linear regression was carried out for each season separately.

RESULTS

The two groups of children were comparable for the following features: age, male-female ratio; the duration of asthma; the occurrence, during the preceding two weeks, of a respiratory infection, a condition that may have influenced lung function test results; the taking of recent medication; and the percentage with a positive skin prick test to an inhalant allergen, an indication of atopy (Table 1). The two groups did differ,

Table 1—*Comparability of Groups*

Features	Mother Nonsmoker (n = 183)	Mother Smoker (n = 56)	p Value (2-tailed)
All patients*			
Mean age, yr	11.0 ± 0.2	10.9 ± 0.4	0.84
Male:Female ratio	120:63	43:13	0.16
Duration of asthma*	6.7 ± 0.3	7.4 ± 0.5	0.26
Recent respiratory infection*	55 (34%)	20 (38%)	0.74
Recent medication*	50 (28%)	22 (41%)	0.12
Positive skin test	158 (86%)	46 (82%)	0.57
Size of mite reaction†	3.4 ± 0.3	1.7 ± 0.4	0.00
New patients*	n = 117	n = 37	
Family history of asthma*	39 (42%)	9 (30%)	0.34
Heating			
Hot air	73 (62%)	20 (54%)	0.48
Wood stove*	14 (12%)	6 (17%)	0.68
Gas for cooking*	7 (6%)	3 (8%)	0.96
Household pets	50 (43%)	22 (60%)	0.22

*When the informant did not know a particular item of information, the patient was omitted from the analysis for that item. The numbers omitted were as follows:
Mother's smoking status, 1; duration of asthma, 33; recent respiratory infection, 84; recent medication, 9; family history of asthma, 32; wood stove, 4; and gas for cooking, 3.
†The size of the mite reaction was not measured in 5. Mean ± SE are presented.

however, in the mean diameter of the wheal produced by a skin prick test with house dust mite extract. Children of nonsmokers had the larger reaction, indicating either that they had a potential for being more severely affected during the cold, wet season, the period when mite-sensitive subjects in Vancouver tend to have the worse asthma,[28] or that they were more highly exposed to house dust mites,[27] or both.

In the 155 patients who were visiting the Allergy Clinic for the first time and were asked the additional questions at that visit, the children of smoking and nonsmoking mothers were also comparable for the following variables: for the percentage of houses in which airborne allergens were circulated by a forced air heating system; the percentage exposed to emissions from gas stoves, used for cooking,[29] or from wood stoves, used for heating;[30] ownership of pets to which they may have been allergic; and the proportion who had a parent or sibling with asthma (Table 1).

This newly seen group of 155 subjects were comparable in all respects to the group of 85 whose first visit to the clinic had been before the start of the study, except that the latter were older by a mean of two years and had had their asthma for one year longer (Table 2).

As in our previous study,[17] there was a highly significant association between maternal smoking and indications of increased asthma severity in the patient. Children of smoking mothers had a lower mean $FEV_1\%$ and FEF25-75%, and had a lower mean PC_{20} histamine

2023380678

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

Table 2—*Comparability Between Old and New Subjects*

Features	Old (n = 85)	New (n = 155)	p Value (2-tailed)
Mean age, yr	12.0 ± 0.3	10 ± 0.2	0.00
Male:Female ratio	56:27	106:49	1.00
Duration of asthma, yr*	7.6 ± 0.5	6.5 ± 0.3	0.06
Recent respiratory infection*	32 (40%)	43 (32%)	0.27
Recent medication*	31 (37%)	42 (29%)	0.24
Positive skin test	75 (88%)	130 (84%)	0.47
Size of mite reaction†	3.0 ± 0.3	3.1 ± 0.3	0.74
FEV_1%	83 ± 1.8	83 ± 1.4	0.91
FEF25-75%	66 ± 2.7	70 ± 2.3	0.47
PC_{20} histamine‡	1.49 ± 1.2	1.82 ± 1.2	0.52

*When the informant did not know a particular item of information the patient was omitted from the analysis for that item. The numbers omitted were as follows: duration of asthma, 33; recent respiratory infection, 24; recent medication, 9.

†The size of the mite reaction was not measured in five.

‡The PC_{20} was performed on 104 subjects (geometric means are given). Mean ± SE are presented.

than did the children of nonsmoking mothers (Table 3).

There was also a significant correlation between the logarithm of the number of cigarettes the mother smoked while in the home and the FEV_1%, the FEF25-75%, and the logarithm of the PC_{20} histamine, suggesting a dose response (Table 4).

Also, as reported in our previous study,[17] there was no association between the father being a smoker or a nonsmoker and the results of any of the above-mentioned tests (Table 3), nor did the number of cigarettes he was said to smoke at home correlate with any test results (Table 4). However, in the subgroup in which the father himself had verified the number of cigarettes he smoked while in the house, there was a correlation between the logarithm of this number and the logarithm of the PC_{20} histamine ($r = -.60$, $n = 9$, $p = 0.045$). This observation suggests that cigarette smoke from the father also increases bronchial irritability in his child but that in the whole group the number of cigarettes smoked by the father was less accurately reported than the number smoked by the mother. A more accurate number for the mother is to be expected, as the history was provided by her alone in 71 percent, by the father alone in 8 percent, and by both together in 18 percent of participants. The disproportionate frequency with which the child was accompanied only by the mother suggested another reason for the good correlation between asthma severity and maternal but not paternal smoking. The mother spent more time than the father in caring for the child, and the child was therefore more intimately exposed to her cigarette smoke. Yet another possible reason for a lesser effect of the father's smoking habits was that he smoked fewer cigarettes (eight per day) when in the house than did the mother (ten per day).

Table 3—*Results of Lung Function Tests Classified by Smoking Habits of Parents*

Feature	FEV_1%	FEF25-75%	PC_{20}† Geometric Mean
Mother*			
Nonsmoker (n = 183)	85 ± 1.2	73 ± 2.0	2.03 ± 1.1 (n = 78)
Smoker (n = 56)	76 ± 2.4	59 ± 3.4	0.91 ± 1.3
p value, two-tailed	0.00	0.00	0.01
Father*			
Nonsmoker (n = 166)	84 ± 1.1	71 ± 2.0	1.90 ± 1.19 (n = 70)
Smoker (n = 86)	81 ± 2.3	66 ± 3.3	1.37 ± 1.20 (n = 34)
p value, two-tailed	0.21	0.46	0.14

*Information about smoking was available for 239 mothers and 232 fathers.

†The PC_{20} was performed on all 104 children who were eligible for the test. *T* tests were carried out on logarithm of the PC_{20} values.

Table 4—*Correlation Between Indicators of Asthma Severity and Log of the Number of Cigarettes Smoked in the House by the Parents, and Probability (p) of r = 0*

Feature	Recent Bronchodilators*	FEV_1%	FEF25-75%	PC_{20}†
Mother‡				
r	0.12	−0.27	−0.27	−0.27
n	226	237	237	104
p	0.04	0.00	0.00	0.00
Father‡				
r	−0.05	−0.06	−0.03	0.00
n	223	232	232	104
p	0.22	0.17	0.33	0.07

*Information about bronchodilators was not known in 9 (point Biserial correlation coefficient).

†The PC_{20} was performed on all 104 children who were eligible for the test. *T* tests were carried out on logarithm of the PC_{20} values.

‡The number of cigarettes smoked by the mother was available in 237 patients and by the father in 232.

As may be expected, if passive smoking aggravates asthma, the children of smoking mothers were more severely affected in the cold, wet season than in the warm, dry season. In the cold, wet season the FEV_1% was 14 percent lower in the children of smokers than in those of nonsmokers, and the FEF25-75% was 25 percent lower (Fig 2 and 3, Table 5). These differences were statistically highly significant. In children seen during the warm, dry season, by contrast, the FEV_1% and FEF25-75% were no lower in children of smokers than in those of nonsmokers. As predicted, spirometric test results were lower if children of smokers were seen in the cold, wet season than if seen in the warm, dry season: the mean FEV_1 was 15 percent lower (t value, −2.23, two tail probability, 0.03), and the FEF25-75% was 23 percent lower (t value, −1.94, two-tail probability, 0.057) (Table 5). A smaller number of children had data for PC_{20} and for recent medication,

2023380679

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

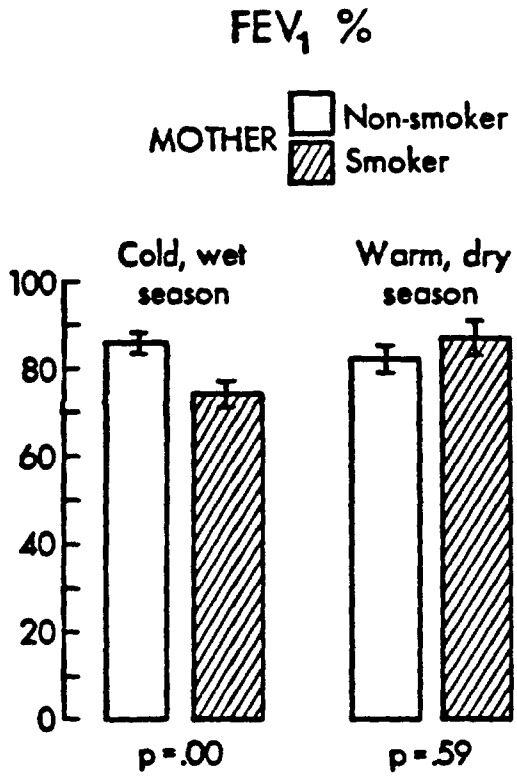

FIGURE 2. The mean FEV_1% predicted in four groups of asthmatic children who were classified by their mother's smoking habits and the season in which they were seen.

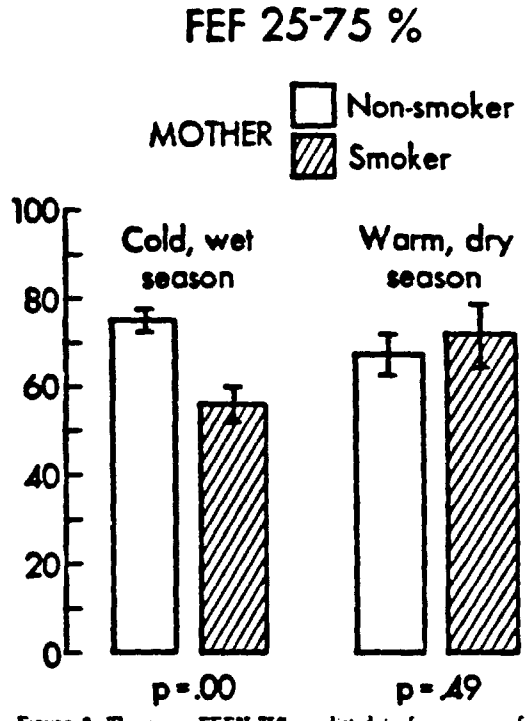

FIGURE 3. The mean FEF25-75% predicted in four groups of asthmatic children who were classified by their mother's smoking habits and the season in which they were seen.

but for these there was nonetheless a significant difference between children of smokers and nonsmokers, if seen in the cold, wet season. In this season the differences also tended to be greater than those observed in the warm, dry season (Tables 5 and 6).

As well, there was evidence of a dose-response to cigarette smoke during the cold and wet but not during the warm and dry months. In the cold, wet season there was a significant correlation between the log of the number of cigarettes the mother smoked while in the house and the likelihood of recent bronchodilator use, and also the extent of the decrease in FEV_1%, in

Table 5—*The Difference Between Indicators of Asthma Severity in Children of Nonsmoking and Smoking Mothers When Measured in the Cold, Wet Season (October-May), Compared With the Warm, Dry Season (June-September)**

Season	FEV,%	FEF25-75%	Geometric Mean PC_{20}
October to May			
Mother nonsmoker (n = 141)	86 ± 1.2	75 ± 2.2	1.95 ± 1.18 (n = 61)
Mother smoker (n = 45)	74 ± 2.7	56 ± 3.8	0.85 ± 1.36 (n = 21)
t value	4.50	4.28	2.53
p (2-tail)	0.00	0.00	0.01
June to September			
Mother nonsmoker (n = 42)	82 ± 2.9	67 ± 4.5	2.37 ± 1.3 (n = 17)
Mother smoker (n = 11)	87 ± 3.6	72 ± 7.4	1.24 ± 2.1 (n = 5)
t value	−0.7	−0.55	1.02
p (2 tail)	0.49	0.59	0.32

*One subject was omitted from the analysis because the smoking status of the mother was not known. T tests were carried out on the logarithm of the PC_{20} values. Mean ± standard errors are presented.

2023380680

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

Table 6—*The Seasonal Differences Between Nonsmoking Mothers' Children and Smoking Mothers' Children as to Whether They Had Taken Bronchodilator Medication Recently*

Season	Recent Bronchodilator Medication* Yes No.	Yes %	No No.	No %
October to May†				
Mother nonsmoker	34	25	102	75
Mother smoker	19	43	25	57
June to September‡				
Mother nonsmoker	16	40	24	60
Mother smoker	3	30	7	70

*Nine subjects were omitted from the analysis because it was not known whether they had taken bronchodilator medication recently.
†χ^2 4.45 (dif = 1) p value (2-tailed) = 0.03.
‡χ^2 0.05 (dif = 1) p value (2-tailed) = 0.83.

FEF25-75%, and in the logarithm of the PC_{20} (Table 7). But during the warm, dry season there was no correlation between the number of cigarettes she smoked and any of these variables.

When multiple regression using FEV_1, FEF25-75%, and log PC_{20} as the dependent variables was carried out for each season separately, the results of the univariate analysis were confirmed. The independent variables were sex, recent respiratory infection, recent medication, positive skin test, family history of asthma, hot air heating, wood stove, gas range, presence of household pets, mother's smoking habits, father's smoking habits, and the logarithm of age, duration of asthma, and number of siblings. In the cold, wet season the number of cigarettes that the mother smoked in the house was the most strongly predictive variable for all three measures of lung function. It was the only variable significantly related to FEF25-75% and to log PC_{20}. But in the case of FEV_1 recent medication also had a significant effect. Conversely, in the warm, dry season, the number of cigarettes the mother smoked in the house had no significant relationship with any of the three measures of lung function. For FEV_1, having a gas range was the only significant predictor but, for FEF25-75%, the duration of the child's asthma and having a gas range were equally strongly correlated with the dependent variable. None of the variables was significant predictors of log PC_{20}.

Table 7—*Correlation Between Indicators of Asthma Severity When Children Are Seen in the Cold, Wet Season Compared With the Warm, Dry Season, and the Logarithm of the Number of Cigarettes Smoked in the House by the Mother, and Probability (p) of r = 0*

Season	Recent Bronchodilators*†	FEV_1%†	FEF25-75%†	PC_{20}‡
October-May				
r	0.17	−0.37	−0.35	−0.28
n	179	185	185	82
p	0.01	.00	.00	<.01
June-Sept				
r	−.08	.08	.04	−.23
n	49	52	52	22
p	.29	.28	.40	.15

*Nine subjects were omitted from the analysis because the respondent did not know whether they had received bronchodilators recently (Point Biserial correlation coefficient).
†Three subjects were omitted from this analysis because the respondent did not know how many cigarettes the mother smoked while in the home.
‡Logarithm of PC_{20} used for correlation.

Discussion

The results suggest that passive smoking in the home worsens bronchial irritability and narrows the bronchi in asthmatic children. These changes were more marked in the children of mothers who smoked, compared with those of mothers who did not smoke, as reported in our previous study.[17] The likelihood that it was passive smoking that caused the increased severity of asthma in the children of smokers, rather than some other difference between them and the children of nonsmokers, was strengthened by the finding that the FEV_1% and FEF25-75% in children of smokers were lower only in the cold, wet season, the period when children are kept indoors, when energy is conserved by closing windows,[22,23] and when concentrations of smoke in the house reach high levels because of decreased ventilation rates.[23-25] During the cold, wet season there was also a significant correlation between the number of cigarettes the mother smoked in the house and the extent of the decrease in FEV_1%, FEF25-75%, and log PC_{20} (Table 7), indicating a dose-response relationship.

In the warm, dry season, by contrast, when children play outdoors and houses are well ventilated, spirometric test results were no lower in children of smokers than in those of nonsmokers, and in smokers' children the mean FEV_1 was higher in those seen in the warm, dry season than in those seen in the cold, wet months. Also, there was no correlation between the number of cigarettes the mother smoked in the house and any lung function test performed during the warm, dry season.

Although the numbers of data on bronchodilator use and PC_{20} are too small for conclusions, they indicate the same trend—that children of smokers have more severe asthma in the cold, wet season than in the warm, dry season.

In our study[17] as in several others, there was little effect attributable to cigarette smoke from the father.[2,3] This finding suggests that children inhale less smoke from their fathers' cigarettes than from their mothers', an explanation supported by the results of a study by

2023380681

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

Jarvis and colleagues.[18] They reported that the mean cotinine level in the saliva of children is 1.95 ng/ml if only the mother smokes and 1.31 ng/ml if only the father smokes. Our study offers three possible reasons for maternal smoking being the more important. One is that fathers, on average, smoke fewer cigarettes in the house than mothers do. Another is that children are more intimately exposed to their mothers and to their cigarettes than to their fathers; the mother alone accompanied 71 percent of our subjects. Third, the number of cigarettes smoked in the house was less accurately reported for the father than for the mother; only the mother was present to provide the history in 71 percent of visits. Kolonel et al[20] found that a wife's estimate of the number of cigarettes her husband smokes is inaccurate: in only 54 percent of cases does it agree within five cigarettes per day with the husband's own estimate. It is, however, unlikely that mothers or fathers were incorrectly classified as smokers or nonsmokers. Kolonel et al[20] reported complete agreement in 95 percent of couples when the man and his wife are asked, separately, whether he is a smoker. A personal history of being a smoker or nonsmoker has also been shown to be accurate, using expired carbon monoxide and serum thiocyanate levels as markers of smoking,[21] nor is it likely that a significant number of the children were incorrectly classified as nonsmokers.

To attribute the results of our study to an error of classification, one would have to postulate that nonsmokers' children are more honest than are smokers' children, and that smokers' children who are seen in the warm, dry season are more honest than are smokers' children seen in the cold, wet season. A more likely explanation for the low prevalence of active smokers in our patient population is that they are aware that cigarette smoke aggravates their asthma, and so they do not smoke. In support of this hypothesis is the finding by O'Connor et al[22] that none of the asthmatic children in their survey was a smoker. When asked privately whether they are smokers, children usually give an honest answer. Pedersen et al[23] measured CO in expired air and found that 5.5 percent, at most, gave false information. Neither is it likely that a difference in social class or referral pattern accounted for the more severe asthma found in children of smoking mothers. Social class is usually determined by the father's occupation,[24] yet we found no relationship between the father's smoking habits and his child's spirometric test results, whereas there was a highly significant relationship between these results and the mother's smoking habits. If smoking mothers are more reluctant to take their children to a pediatric allergist than nonsmoking mothers, one would expect children of smokers to have had asthma for a greater number of years. This was not the case. There was no significant difference between the two groups in the duration of the child's asthma.

Although there was no association between the father's smoking and the results of the children's spirometric tests, there was nonetheless some evidence that his smoke might be influencing bronchial irritability in the child. There was a significant correlation between the logarithm of the child's PC_{20} and the logarithm of the number of cigarettes the father himself said he smoked while in the house.

Greater exposure to other pollutants, such as combustion products from wood-burning stoves[25] or gas for cooking,[26] did not seem to account for the worsened asthma in the children of smoking mothers. Similar proportions in both groups used wood stoves for heating and gas stoves for cooking. Nor was there a significant difference between the two groups in ownership of pets, which might aggravate asthma in susceptible subjects, nor in the use of forced-air heating systems, which might circulate allergens.

Our demonstration that passive smoking affects asthma significantly only during the cold, wet season is consistent with results obtained in surveys of representative samples of schoolchildren in different regions of North America and in Britain. Those surveyed in Arizona, a warm, dry part of the United States,[7,9] and those surveyed during the summer in Britain[28] showed no significant difference in spirometric test results between children of smokers and nonsmokers. Surveys conducted in areas with cold winters, by contrast, usually reveal significant differences between the two groups.[1-6] But even in these areas, the difference in spirometric values between children of smoking mothers and nonsmoking mothers is small, not exceeding 5 percent, and is considerably smaller than in our study on asthmatic subjects, suggesting that representative groups of schoolchildren are less severely affected by maternal smoking than are asthmatic children.

Our finding that bronchial responsiveness is greater in asthmatic children of mothers who smoke than in those of mothers who do not smoke is also consistent with previously published articles. We reported earlier an increased bronchial responsiveness in children of smoking mothers in a group of 94 asthmatic children;[17] such an increase was also subsequently noted in a community-based sample of 21 asthmatic children by O'Connor et al.[22]

Passive smoking is likely to be the cause of the greater bronchial irritability and the increased bronchial obstruction which we find in asthmatic children of smoking mothers compared with those of nonsmoking mothers. Although children in the two groups are comparable in other respects and have similar indications of asthma severity during the warm, dry season when their houses are well ventilated, children of

2023380682

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000

smoking mothers have pulmonary function that is significantly poorer during the cold, wet season when houses are closed up, when indoor cigarette smoke reaches its highest level, and when the children spend more time in the house.

ACKNOWLEDGMENTS The authors thank Mr. Ronnie Sizto for computer programming, Mrs. Janice Fuller, R.N., for interviewing patients, and Mrs. Huguette Brown for performing the tests.

REFERENCES

1 Weiss ST, Tager IB, Speizer FE, Rosner B. Persistent wheeze: its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. Am Rev Respir Dis 1980; 122:697-707

2 Hasselblad V, Humble CG, Graham MG, Anderson HS. Indoor environmental determinants of lung function in children. Am Rev Respir Dis 1981; 123:479-85

3 Vedal S, Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease: analysis of pulmonary function. Am Rev Respir Dis 1984; 130:157-92

4 Letwitter FJ, Tager IB, McGue M, Tishler PV, Speizer FE. Genetic and environmental determinants of level of pulmonary function. Am J Epidemiol 1984; 120:518-29

5 Ware JH, Dockery DW, Spiro A, Speizer FE, Ferris BG. Passive smoking, gas cooking and respiratory health of children living in six cities. Am Rev Respir Dis 1984; 129:366-74

6 Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Higgins ITT. Passive smoking in childhood: respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis 1986; 133:966-73

7 Lebowitz MD, Burrows B. Respiratory symptoms related to smoking habits of family adults. Chest 1976; 69:48-50

8 Dodge R. The effects of indoor pollution on Arizona children. Arch Environ Health 1982; 37:151-55

9 Lebowitz MD. The effect of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic and non-asthmatic families. Eur J Respir Dis (suppl) 1984; 133:90-97

10 Weiss ST, Tager IB, Schenker M, Spiezer FE. The health effects of involuntary smoking. Am Rev Respir Dis 1983; 125:933-42

11 Aronow WS. Effect of passive smoking on angina pectoris. N Engl J Med 1978; 299:21-24

12 Lefcoe NM, Ashley MJ, Pederson LL, Keays JJ. The health risk of passive smoking: the growing case for control measures in enclosed environments. Chest 1983; 84:90-95

13 Smoking and health: a report of the Surgeon General. Publication no. 79,50066. Washington DC: Department of Health, Education and Welfare, 1979

14 Dockery DW, Spengler JD. Indoor-outdoor relationships of respirable sulfates and particles. Atmos Environ 1981; 15:335-43

15 Repace JL, Lowry AH. Indoor air pollution, tobacco smoke, and public health. Science 1980; 208:464-72

16 Climate normals. Vol 4. Humidity. Toronto: Canada Dept of Transport, 1968:20

17 Murray AB, Morrison BJ. The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma. J Allergy Clin Immunol 1986; 77:575-81

18 Jarvis MJ, Russell MAH, Feyerabend C, Eiser JR, Morgan M, et al. Passive exposure to tobacco smoke: saliva cotinine concentrations in a representative population sample of non-smoking school children. Br Med J 1985; 291:927-29

19 Ferris BJ. Epidemiology standardization project. Am Rev Respir Dis 1978; 118(pt 2):1-118

20 Murray AB, Ferguson AC, Morrison BJ. Airway responsiveness to histamine as a test of overall severity of asthma in children. J Allergy Clin Immunol 1981; 66:119-24

21 Polgar G, Promadhat V. Standard values in pulmonary function testing in children. Philadelphia: WB Saunders Co, 1971:179

22 Cockcroft DW, Killian DN, Mellon JJA, Hargreave FE. Bronchial reactivity to inhaled histamine: a method and clinical survey. Clin Allergy 1977; 7:235-43

23 Murray AB, Simons E, Ferguson AC, Freigang B, Gerrard JW, et al. Skin testing for allergy in children. Can Med Assoc J 1983; 129:826-30

24 Canadian climate normals. Vol 4. Degree days, 1951-1960. Atmospheric Environment Service, Environment Canada 1982:41

25 Korsgaard J. House dust mites and absolute indoor humidity. Allergy 1983; 38:85-92

26 Murray AB, Ferguson AC, Morrison BJ. Diagnosis of house dust mite allergy in asthmatic children: what constitutes a positive history? J Allergy Clin Immunol 1983; 71:21-25

27 Murray AB, Ferguson AC, Morrison BJ. Sensitization to house dust mites in different climatic areas. J Allergy Clin Immunol 1985; 75:106-12

28 Speizer FE, Ferris B, Bishop YMM, Spengler J. Respiratory disease rates and pulmonary function in children associated with NO_2 exposure. Am Rev Respir Dis 1980; 121:3-10

29 Honicky RE, Osborne JS, Akpom CA. Symptoms of respiratory illness in young children and the use of woodburning stoves for indoor heating. Pediatrics 1985; 75:567-93

30 Kolonel L, Horohata T, Nomura A. Adequacy of survey data collected from substitute respondents. Am J Epidemiol 1977; 106:476-84

31 Vogt T, Selvin S, Widdowson G, Hulley S. Expired air carbon monoxide and serum thiocyanate as objective measures of cigarette exposure. Am J Public Health 1977; 67:545-49

32 O'Connor GT, Weiss ST, Tager IB, Speizer FE. The effect of passive smoking on pulmonary function and nonspecific bronchial responsiveness in a population-based sample of children and young adults. Am Rev Respir Dis 1987; 135:800-04

33 Pedersen L, Sidney K, Lefcoe N. An objective measure of the validity of children's responses to a questionnaire on health and smoking. Can J Public Health 1977; 68:497-98

34 Blishen BR. The construction and use of an occupational class scale. Can J Econ Political Sci 1956; 24:519

35 Yarnell JWG, St Leger AS. Respiratory illness, maternal smoking habits, and lung function in children. Br J Dis Chest 1979; 73:230-36

2023380683

Source: https://www.industrydocuments.ucsf.edu/docs/mynx0000